Effect of Small-World Networks on Epidemic Propagation and Intervention

Zengwang Xu,1 Daniel Z. Sui2
1Houston Advanced Research Center, The Woodlands, TX, 2Department of Geography, Texas A&M University, College Station, TX

The small-world network, characterized by special structural properties of high connectivity and clustering, is one of the highlights in recent advances in network science and has the potential to model a variety of social contact networks. In an attempt to better understand how these structural properties of small-world networks affect epidemic propagation and intervention, this article uses an agent-based approach to investigate the interplay between an epidemic process and its underlying network structure. Small-world networks are derived from a network “rewiring” process, which readjusts edges in a completely regular two-dimensional network by different rewiring probabilities (0–1) to produce a spectrum of modified networks on which an agent-based simulation of epidemic propagation can be conducted. A comparison of simulated epidemics discloses the effect of small-world networks on epidemic propagation as well as the effectiveness of different intervention strategies, including mass vaccination, acquaintance vaccination, targeted vaccination, and contact tracing. Epidemics taking place on small-world networks tend to reach large-scale epidemic peaks within a short time period. Among the four intervention strategies tested, only one strategy—the targeted vaccination—proves to be effective for containing epidemics, a finding supported by a simulation of the severe acute respiratory syndrome epidemic in a large-scale realistic social contact network in Portland, OR.

Introduction

Throughout human history, emerging infectious diseases have been demonstrating their ability to spread rapidly through social contacts and pose severe threats to the whole world as shown by the outbreak of the severe acute respiratory syndrome (SARS) epidemic in 2003. Human social contacts can be modeled by network structures in which individuals are vertices and social contacts between individuals are edges. In fact, network approach for modeling disease propagation and intervention in social contact networks has emerged as a promising modeling strategy

Correspondence: Zengwang Xu, Houston Advanced Research Center, 4800 Research Forest Dr., The Woodlands, TX 77381
e-mail: zxu@harc.edu

with a great potential to tackle the complexity in disease propagation (Newman 2002; Bian 2004, 2007; Jeger et al. 2007; Riley 2007). However, the effect of network topology on epidemic propagation for informing basic choice of topology in modeling the diffusion of epidemics is still not very well understood (Riley 2007). Particularly, recent studies in network science show that a small-world network model has the potential to model the social contact network for disease control (Liljeros et al. 2001; Newman 2002), but no further empirical studies have been reported. To better understand the effect of network topology on epidemic diffusion in general and the potential of the small-world network model on epidemic modeling in particular, this article uses an agent-based simulation to investigate the effect of properties of small-world network structure on epidemic diffusion and the effectiveness of intervention strategies. By doing so, we aim to fill this void in the literature.

Networks have been considered a ubiquitous underlying structure for many real-world complex systems, such as transportation, computer, and social contact networks. Recent years have witnessed voluminous research on structural properties of complex networks and dynamics occurring on the complex networks, and new syntheses have been made to promote the “new” science of networks in which the small-world network is one of the major advances (Watts 1999, 2004; Strogatz 2001; Barabasi 2002; Dorogovstev and Mendez 2003; Newman, Barabasi, and Watts 2006). The so-called small-world network was named after the small-world phenomenon, popularly known as the “six degrees of separation” between any random pair of strangers in social contact networks. The study on the small-world problem dates back to 1967, when Milgram (1967) conducted the groundbreaking, empirical sociological study that the small-world phenomenon exists in human contact networks. Watts and Strogatz (1998) identified the first small-world network model, which exists along a spectrum of networks “rewired” between completely random and completely regular network models, and has both the high clustering of regular networks and the high connectivity of random networks. Their finding was followed by a surge of studies on small-world networks, particularly experimental studies, which show that small-world network is a ubiquitous underlying structure existing in a variety of real-world networks including social, information, technological, and biological networks (Watts 1999, 2004; Barabasi 2002; Newman 2003). As the structure of networks always affects dynamics occurring on the networks, the interplay between dynamics taking place on complex networks and the structural properties of complex networks has been considered as one of the mainstream studies on complex networks (Newman 2003). The objective of this article is to investigate the interplay between the structure of small-world network and epidemic propagation through networks as well as effective intervention strategies, and it aims to provide a better understanding on the applicability of the small-world network model on epidemic modeling and intervention strategies.

The second section of this article reviews two major epidemiological models (the compartment model and the network model) and control strategies for
epidemics. The third section describes the methodology of this study, including the epidemic process, the emergence of small-world networks, and the simulation of an epidemic on networks. Simulation results regarding epidemic diffusion and control strategies are presented in the fourth section. The last section contains a summary and conclusions.

**The network modeling of epidemic propagation and intervention strategies**

Two major types of epidemic models—compartmental and network models—have been developed to predict epidemic propagation and inform possible intervention strategies (Keeling 2005). Compartment models classify the population into several epidemiological compartments, such as S (susceptible), E (exposed), I (infective), and R (recovered). People are all susceptible before the first infective is introduced. After being infected, individuals of susceptible class can enter the exposed class E of those in the latent period, who are infected but not yet infectious. After the latent period ends, individuals in class E enter class I of infective who are capable of transmitting the infection. Infectious individuals could be removed by death or entering the recovered class R consisting of those with permanent infection-acquired immunity (Hethcote 2000). This epidemic process is also named the SEIR model. For different epidemics, the SEIR model has a few variants, such as the SIR and SIS models.

With compartment models, it is possible to apply nonlinear differential equations to model the temporal dynamics of disease propagation (Hethcote 2000). However, compartmental models assume, rather unrealistically, that everybody is equally likely to infect all other susceptible individuals (Hethcote 2000; Newman 2002). In fact, the infectious diseases diffuse through human populations only along contact between infective (those who were infected and are able to infect others) and susceptible (those who are susceptible to be infected), and an infective can only infect those he/she has contacts with, but not anybody else. Moreover, compartment models ignore the pattern of the disease transmission, which is crucial for the study of intervention strategies.

Epidemic modeling has been experiencing a change from deterministic to stochastic and from compartmental to network paradigms in recent year (Moore and Newman 2000; Koopman 2003; Zheng et al. 2005). Aiming to increase realistic heterogeneity, recently reported studies on network modeling of epidemics have modified traditional quantitative models in two aspects: (1) replacing the homogeneity assumption by a network of connections among individuals and (2) considering different probabilities of infection on the connections (Pastor-Satorras and Vespignani 2001; Newman 2002; Pastor-Satorras and Vespignani 2002). The importance of network structure for disease propagation has long been recognized among researchers (Wallinga, Edmunds, and Kretzschmar 1999; Lloyd and May 2001; Read and Keeling 2003; Bian 2004, 2007). However, the existing regular and
random network models are not compatible with the models based on social contact networks, because the structure of social networks is neither completely random nor completely regular. As aforementioned, the small-world network model captures the important features of social contact networks. Although there are many different kinds of social networks depending on what constitutes the social links, the small-world structure is a common feature of them, such as human interaction networks (Milgram 1967), human sexual contact networks (Liljeros et al. 2001; Eames and Keeling 2002), and scientific collaboration networks (Newman 2001a, b, c; Newman 2004). Advances in network science in recent years have provided overwhelming evidence that the small-world network model is a promising approach for modeling the real-world social contact networks.

Because of its capability to take into account realistic heterogeneity in transmission patterns, the small-world network model also offers us an innovative way to examine the effectiveness of different intervention strategies in combating epidemics, such as different vaccination, quarantine strategies, and controlling infections at the source (Longini et al. 2005). Typically, vaccine development is only the first step to combat any epidemics of global threats. Furthermore, even when there exists enough vaccine, it still needs an improved understanding on how to conduct the vaccination process optimally (Anderson and May 1982, 1985). It is now widely agreed that adopting appropriate strategies could dramatically increase efficiency in epidemic intervention (Eames and Keeling 2003). The goal of all intervention strategies is to reduce the morbidity and mortality through reducing the number of susceptible (by vaccination) and/or restricting transmission (by quarantine or isolation). To achieve this goal, four generic vaccination and intervention strategies have been proposed in the literature: mass (Kaplan, Craft, and Wein 2002), targeted (Huerta and Tsimring 2002), traced (Eames and Keeling 2003; Tsimring and Huerta 2003; Kiss, Green, and Kao 2005), and acquaintance vaccinations (Pastor-Satorras and Vespignani 2002; Madar et al. 2004).

A mass vaccination strategy is theoretically capable of preventing the diffusion of an epidemic, and it was used successfully in the eradication of smallpox in the late 1970s (Kaplan, Craft, and Wein 2002). However, it is not practical economically to conduct a mass vaccination, and its adverse effects to certain population groups could be severe (Ferguson et al. 2003). Furthermore, the mutation of certain pathogens could be more rapid than the mass vaccination can follow. However, among other strategies, we still consider mass vaccination a possible option. In network models, mass vaccination is implemented by random immunization of a certain proportion of the vertices. The targeted vaccination is to immunize the highest connected vertices for a certain amount (Pastor-Satorras and Vespignani 2002). We adopt the same concept to immunize the highest connected vertices until the desired proportion of population is reached. Traced vaccination or contact tracing strategy (Huerta and Tsimring 2002; Eames and Keeling 2003; Tsimring and Huerta 2003; Kiss, Green, and Kao 2005; Keeling and Eames 2005) traces the contacts of infected individuals and immunizes their connected vertices. For the
contact tracing strategy in this study, infected vertices and their connected vertices are traced and controlled for a period of time, and if the connected vertices are infective, the tracing will continue until all susceptible neighbors are found. During the tracing period, the traced vertices will not be able to infect others. After the traced period, controlled vertices change to recovered if they were infective before tracing and change back to susceptible if they were susceptible before tracing. Acquaintance vaccination was originally designed to select a random fraction of the vertices and look for a random acquaintance for each chosen vertex to immunize (Madar et al. 2004). We adopt the same idea in acquaintance vaccination to select a random fraction of the vertices and immunize their immediate neighbors until the expected vaccination proportion is reached. This strategy is more likely to select and vaccinate highly connected vertices without knowing the full knowledge of network as in the targeted strategy.

**Methodology**

**The small-world network model for social contact networks**

Social contacts in this article are represented by networks in which the vertices represent individuals and the edges represent contacts between individuals. As aforementioned, the small-world network model is considered a potential model for social contact networks and it was initially discovered through a network rewiring process (Watts and Strogatz 1998). The network rewiring process attempts to introduce randomness to a regular network, and for every vertex in the network, it readjusts each of its edges to another randomly selected vertex by certain probability (0–1). When the rewiring probability is 0, the rewiring process will not readjust any edges of the regular network and the readjusted network will remain a regular network. When the rewiring probability is 1, the rewiring process will readjust every edge and the readjusted network will be a completely random network. When the rewiring probability is between 0 and 1, the rewiring process will readjust every edge according to the rewiring probability and the readjusted network will be a network with certain randomness quantified by the rewiring probability. With a series of consecutively increasing rewiring probabilities from 0 to 1, for example, 0.0001, 0.0005, 0.001, 0.005, 0.01, 0.05, 0.1, 0.9, and 1, the regular network will be rewired (readjusted) to a spectrum of networks that are interpolated between completely regular and completely random networks. Although the first small-world network model (Watts and Strogatz 1998) was discovered from rewiring a one-dimensional (1D) network (a ring lattice network), the same rewiring process can be applied to networks in a 2D context.

In this article, the small-world networks are obtained by rewiring a regular 2D lattice using the same rewiring process used by Watts and Strogatz (1998). The rewiring process generates a spectrum of networks with consecutively increasing rewiring probabilities from 0 to 1, in which small-world networks are in the intermediate range between 0 and 1. In the rewiring process, two measurements, the
average path length (APL) and the clustering coefficient (CC), are used to monitor the change of structural properties of networks as well as the emergence of small-world networks. Using the same definitions of Watts and Strogatz (1998), the APL is the average length of the shortest path (number of edges on the shortest path between two vertices) between any two vertices over the network. The CC of a vertex is the ratio between the existing edges and all possible edges among its connected neighbors. The CC of the network is the average of the CC of all vertices in the network. Conceptually, the APL is a measure of the overall connectivity and CC of a network is a measure of overall clustering of the network. As aforementioned, the rewiring process starts from a regular 2D lattice network with 100 vertices, each one connected to its eight nearest neighbors, except the vertices along the boundary having five nearest neighbors and the vertices at four corners having only three nearest neighbors. For every vertex, all its edges are rewired according to a probability \( f(0 - 1) \) to a randomly chosen vertex, and a shortcut link is added only if there is not an edge between them. The rewiring process is able to generate a spectrum of networks with different rewiring probabilities between 0 and 1. Fig. 1 shows schematically the spectrum of networks using a 10 × 10 completely regular network, a small-world network, and a completely random network. Fig. 2 shows the variation of the APL and CC of networks rewired by different probabilities (0–1) during the rewiring process. Consistent with Watt and Strogatz’s (1998) results, small-world networks emerge in an intermediate range of rewiring probabilities between 0 and 1 when the APL drops dramatically while the CC remains very high (shaded range in Fig. 2). This spectrum of networks actually illustrates the possible topological connections evolving from completely regular to complex random.

**Agent-based simulation on networks**

Each vertex in the network is considered as an autonomous agent, and it has five possible statuses: susceptible, infective, recovered, vaccinated, and traced (Sir-
The simulation starts from one randomly selected susceptible vertex and follows the interacting rules below:

1. all the vertices are susceptible or vaccinated before the simulation starts;
2. the infectious vertex can only infect its susceptible connected neighbors with 90% probability;
3. infected vertices become recovered after a period of infection;
4. the traced (controlled) vertices will become recovered if they were infected and susceptible if they were susceptible; and
5. recovered vertices will remain recovered and unable to be infected again for the rest of their life cycle. Fig. 3 depicts schematically the possible change map of the status of a vertex throughout its life cycle.

Our first simulation is an epidemic propagating on the spectrum of networks without applying any intervention strategies. This simulation is also used as a control for the second simulation on different intervention strategies. The epidemic in this simulation is an SIR process, that is, each vertex in the network represents an individual who could have three epidemiological statuses: susceptible ($\gamma(t) = 0$),
infective \((0 \leq \gamma(t) < 12)\), and recovered \((12 \leq \gamma(t))\). Each vertex has a discrete time counter \(\gamma(t)\) to determine how long this vertex has stayed in different epidemiological statuses. Time proceeds by discrete steps, and the discrete time unit in this simulation could be considered as days or hours depending on the epidemiology of the specific disease. As the discrete time step increases, the infected individuals will infect their connected susceptible individuals. In addition, the epidemiological status of each vertex also depends on the status of its neighbors. A susceptible vertex could only be infected by an infectious neighbor. The simulation process stops when the network has no infected individuals. The simulation results are presented in the next section.

Our second simulation is to simulate the same epidemic propagating on the same spectrum of networks, but with different intervention strategies applied to the network before the epidemic starts. It aims to determine the effectiveness of different intervention strategies on the epidemic intervention. In the second simulation, each vertex in the network, beside the three epidemiological statuses (SIR), has two additional statuses to indicate its intervention status, that is, traced (controlled) and vaccinated. The vaccinated intervention status of vertices will be predetermined according to different aforementioned intervention strategies (mass, targeted, and acquaintance vaccination) before the epidemic starts. For the traced vaccination strategy, the detection period and the traced period were arbitrarily set as 5 and 5 days, respectively, which means that the infected vertices will infect others for 5 days until they are detected and once detected they are controlled (traced) for 5 days. These time periods could be changed according to the epidemiology for different diseases, but this will not change the conclusions. The three other intervention strategies are three different ways to select vertices to vaccinate. For each strategy, we vaccinate different percentages of the population (10%, 20%, 30%, 40%, and 50%) through respective intervention strategies, that is, mass, targeted, acquaintance intervention strategies, before any vertices are infected. For each vaccination percentage, for example, 50%, the effectiveness of intervention strategies is pronounced by comparing the epidemic dynamics taking place on networks applied with different intervention strategies (Rhodes and Anderson 1997). The contact tracing strategy will be applied to the network after the infection starts. Therefore, the epidemic diffusion in this study will be simulated not only on a spectrum of rewired networks to learn how structural properties of networks affect epidemic propagation but also on vaccinated networks to investigate the effectiveness of different intervention strategies. The simulation is implemented by a C++ program code according to the methodology described by the flowchart in Fig. 4.

**Simulation results**

**Epidemic diffusion on networks**

The propagation of epidemics on networks is depicted by the infective curve or cumulative infective curve that can be characterized by two properties: maximum
epidemic size (MES) and time to reach MES (TMES) (Ferguson et al. 2003; Shirley and Rushton 2005). Fig. 5 only shows the MES and TMES in infective curve, but they can also be used to characterize cumulative infective curve. The same epidemic process taking place on networks of different topologies produces different (cumulative) infective curves (Fig. 6), therefore different MESs and TMESs. The difference in network topology is characterized by rewiring probability, APL, and CC. Compare and contrast between the epidemic properties (MES and TMES) and network properties (rewiring probability, APL, and CC) provide a basic understanding of how network topology affects epidemic propagation.

The randomness in connection topology of a network, represented by the rewiring probability, severely affects the epidemics taking place on the network. The randomness of networks affects the MES and TMES in different ways: the MES increases and the TMES decreases with the rewiring probability of networks (Fig. 7). When the randomness (or rewiring probability) of the network is zero, MES of the epidemic can only reach 26% of the vertices, that is, the epidemic can only affect at most 26% of the vertices (Fig. 7). The MES reaches 36% when the rewiring probability is 0.0001, and it can reach 85% when the rewiring probability is 0.001. The epidemic can spread to the whole network when the rewiring probability is > 0.01 (Fig. 7). In other words, 1% of randomness will dramatically increase the vulnerability of the network for epidemic propagation. Not only will the introduction of a small amount of randomness in network topology cause a huge increase in the epidemic size, but also the epidemic will take a very short time to reach its large size. The randomness in network topology affects the TMES in a different way. Higher randomness corresponds to smaller TMES. Epidemics have the largest TMES on regular network and smallest TMES on random networks. Epidemics occurring

Figure 4. The flowchart of the simulation of epidemic diffusion on networks.

Effect of Small-World Networks on Epidemic Propagation
Zengwang Xu and Daniel Z. Sui
on small-world networks have already taken a very short time to reach very large epidemic sizes (Fig. 7). In addition, the MES has a clear linear relation with its TMES ($y = -159.77x + 11618$, $R^2 = 0.8575$) (Fig. 8). It implies that the MES inversely corresponds to the TMES in small-world networks.

As aforementioned, two structural properties of networks, connectivity and clustering, are characterized by APL and CC, respectively. These two measures show different relations with the MES and TMES of epidemics (Figs. 9 and 10). The APL has linear relationships with both MES ($MES = -0.8923APL + 1.1257$, $R^2 = 0.9601$) and TMES ($TMES = 0.8667APL + 0.0805$, $R^2 = 0.8757$) (Fig. 9). A high APL (low connectivity) corresponds to a low MES, and a high TMES and a low APL (high connectivity) corresponds to a high MES and a low TMES. It implies that high connectivity usually corresponds to a large epidemic, and the epidemic has a short time to reach its maximum size. Unlike APL, CC has completely different relations with both MES and TMES (Fig. 10). The MES and TMES rapidly reach extreme status when the CC is still very high (e.g., 0.9 or higher); but when CC is lower than 0.9, the corresponding MES and TMES do not change significantly (Fig. 10). The small-
world networks, which are characterized with high CC and low APL, are vulnerable in their topological structure for epidemics spreading with a large MES and small TMES.

**Effectiveness of intervention strategies**

Following Zanette and Kuperman (2002), we use the maximum infection rate to measure and compare the effectiveness of different intervention strategies on the epidemic diffusion. The maximum infection rate is defined as the ratio between the maximum infected population and total susceptible population. A large maximum

![Figure 7](image_url)

**Figure 7.** The variation of maximum epidemic size (MES) and time to reach MES on networks of different rewiring probabilities.

![Figure 8](image_url)

**Figure 8.** The linear relation between maximum epidemic size (MES) and time to reach MES.
infection rate implies that a large proportion of population is infected in the epidemic. The intervention strategy is not effective if the epidemic has a large maximum infection rate.

As shown in Figs. 11a, c, e, and 12, all intervention strategies become less effective with the increasing of the rewiring probability, because the maximum infection rates have dramatic increases for all intervention strategies as the rewiring probability increases. When rewiring probabilities are >0.01, the maximum infection rates could reach 1, implying that all individuals in the network could be infected. We also investigate the sensitivity of intervention strategies on different vaccination percentage. However, we only investigate the vaccination percentage ≤50% (except mass vaccination for 60%), because once larger percentage of vertices are vaccinated, the networks could become segmented for epidemic

Figure 9. The relations between average path length and maximum epidemic size (MES) and time to reach MES.

Figure 10. The relations between clustering coefficient and maximum epidemic size (MES) and time to reach MES.
propagation. For mass vaccination and acquaintance vaccination, the maximum infection rates decrease with the increasing of the vaccination proportion. Targeted vaccination has very similar low infection rates when vaccination proportions are 0.1, 0.2, and 0.3, but it has a huge variation in infection rate when vaccination proportions are 0.4 and 0.5. Because targeted vaccination immunizes highly connected vertices first, a small vaccination rate could be more effective as shown in Figure 11. The variation of the maximum infection rate and time to reach maximum epidemic size (TMES) with rewiring probability under different control strategies: (a)–(b) mass vaccination, (c)–(d) acquaintance vaccination, and (e)–(f) targeted vaccination.
Fig. 11. Comparing Figs. 11a and c, we found that acquaintance vaccination is more effective than mass vaccination, particularly when the vaccination proportion is high, such as 0.5. Moreover, acquaintance vaccination performs better than mass vaccination when the rewiring probability is high, for example, when rewiring probability is 0.1, and for 50% vaccination rate, maximum infection rate is 0.822 for acquaintance vaccination (Fig. 11c), but 0.9772 for mass vaccination (Fig. 11a). The TMES for all the intervention strategies decrease with the increasing of the rewiring probability (Figs. 11b, d, and f). In general, the higher proportion of vaccination corresponds to longer TMES. However, when rewiring probability is very low, TMES may have a large variation.

The maximum infection rate of contact tracing increases dramatically when the rewiring probability increases. The TMES decreases with the increasing of the rewiring probability. Fig. 12 shows the variation of the MES and TMES of contract tracing while increasing the rewiring probability and their comparison with the MES and TMES of the epidemics without any intervention.

In order to study the performance of different intervention strategies (mass vaccination, targeted vaccination, and acquaintance vaccination) on low and high percentages of vaccination, we compare the effectiveness of intervention strategies when 10% and 50% of the population are vaccinated. As shown in Fig. 13a, when 10% of the population is vaccinated, the targeted vaccination is the most effective. Contact tracing is more effective than mass and acquaintance vaccinations. Mass and acquaintance vaccinations show almost identical results; however, the acquaintance vaccination has more variation when the rewiring probability is low. As shown in Fig. 13b, when 50% of the population is vaccinated, acquaintance vaccination is the most effective regardless of whether the networks are rewired by low or high probabilities. Targeted vaccination is most effective when the networks are rewired by medium probabilities. Because completely random
networks and completely regular networks are both homogenous, rewiring networks with either high or low probability tends to make them more homogeneous. Targeted vaccination is not effective on homogenous networks when these networks have low or high rewiring probabilities. Overall, the effectiveness of intervention strategies is affected by the randomness (as indicated by rewiring probability) of the networks and vaccination proportions. Table 1 summarizes the most effective intervention strategies for the networks with different degrees of randomness.

SARS propagation in a realistic urban social network
To further test the effects of network topology on the diffusion of epidemics in a real-world context, we applied the same methodology to examine the SARS propagation in a realistic urban social network. Eubank et al. (2004) generated a large social contact network for Portland, OR, through a large-scale individual-based urban traffic simulation built on actual census, land-use, and population mobility data. The network comprises of 1,515,271 individuals (vertices) and 41,860,740 social contacts among the individuals (edges), and it is a highly connected small-world network (Eubank et al. 2004). We use this network as an empirical social contact network to examine the SARS epidemic propagation and the effectiveness of different intervention strategies.

Table 1  The Effective Intervention Strategies for Networks with Different Vaccination Proportion and Randomness

<table>
<thead>
<tr>
<th>Network randomness (rewiring probability)</th>
<th>Vaccination proportion</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>High (50%)</td>
</tr>
<tr>
<td>High</td>
<td>Acquaintance</td>
</tr>
<tr>
<td>Small-world</td>
<td>Targeted, acquaintance</td>
</tr>
<tr>
<td>Low</td>
<td>Acquaintance</td>
</tr>
</tbody>
</table>
Using the same agent-based simulation methodology described above in the “Agent-based simulation on networks” section, the SARS epidemic starts from a randomly selected individual, spreads along the social contacts, and stops when no individual is infected anymore. To investigate the effectiveness of intervention strategies, we vaccinate different percentage of the population (10%, 20%, 30%, 40%, and 50%) through respective strategies like the mass, acquaintance, and targeted strategies before the epidemic starts.

Mostly, an individual exposed to SARS may become infectious after 2–7 days, and most infectives can recover after 7–10 days. We use an SIR model to represent SARS epidemic process, in which the infectious period is 10 days. In the contact tracing strategy, we suppose an infective can be identified after 5 days’ infection and then controlled. The control period is 7 days. We realize that these epidemiological parameters vary for different individuals in real-world infection. Because our simulation focuses on how the network structure affects epidemic propagation and intervention rather than the epidemic dynamics per se, we assign a fixed number for these parameters based on SARS’ epidemiology, and it suffices for our study.

The simulated SARS epidemic is represented by infective curves (Fig. 14). Without any vaccination, the network is quite vulnerable to an SARS epidemic, and a very large proportion of the population is infected—a conclusion also supported by Meyers et al. (2005). The contact tracing strategy can rapidly slow down and finally control the epidemic, and early detection is essential for its effectiveness. Our simulation use 5 days as the detection period, that is, the infective can infect others for 5 days before they are detected and controlled. This detection period is obviously too long, because the epidemic can infect a very high proportion

![Graph showing SARS epidemic propagation and intervention](Figure 14. SARS epidemic propagation and intervention in the social network with 50% population vaccinated by mass, targeted, or acquaintance intervention strategies.)
of the population. The effectiveness of the other three intervention strategies is consistent with what we have found in our simulation on the hypothetic networks. As aforementioned, we have vaccinated different percentages of the population (10%, 20%, 30%, 40%, and 50%) through each of these strategies. For different vaccination percentages, no matter if it is 10% or 50%, the infective curves for different strategies show the same pattern, and the pattern for 50% vaccination is the most apparent (Fig. 14). Fig. 14 shows the infective curves of SARS when 50% of the population is vaccinated using these three different strategies. The targeted strategy is still the most effective, and it dramatically delays the epidemic peak. The mass vaccination is the least effective. The acquaintance vaccination is somewhere between targeted and mass vaccinations. However, the acquaintance vaccination controls the epidemic peak as effectively as the targeted strategy, but it is not as effective in delaying the epidemic peak. This is largely due to the nosocomial nature of SARS diffusion as demonstrated by Small and Tse (2005) during the very early stage of the SARS epidemic in Hong Kong in 2003.

Summary and discussions

Our study, following and expanding the original methodology by Watts and Stragrotz (1998), has investigated the effect of network structure, particularly the small-world network, on epidemic propagation and intervention in a hypothetical network and a realistic social network derived from empirical data for Portland, OR. We have found that epidemics taking place on the small-world networks are most likely to reach large epidemic sizes in a short time period. Among the four intervention strategies we studied, the targeted strategy is the most effective for small-world networks. These four generic intervention strategies have covered basic elements in the epidemic intervention implemented in the real world. Any of these intervention strategies, on its own, already has a significant impact on the epidemic propagation. In the real world, their implementations may be not as ideal and efficient as in our simulation, but in combination, they can be even more effective. In particular, it may be hard to implement the targeted strategy due to the lack of full knowledge of the real-world network, but the acquaintance strategy is able to provide a mechanism to locate the highly connected targets in real-world networks. This is supported by our simulation for the diffusion of SARS epidemic in Portland, OR.

The infection probability along the network connection is considered to be 90% throughout the networks in our simulation. However, this probability varies a lot in real-world contacts according to each individual's characteristics such as occupation, age, gender, etc. How to simulate and incorporate correctly the variable infection probability in network connection patterns is still an open research question. It is necessary for future simulations on specific epidemic dynamics to incorporate realistic heterogeneity in the infection probability. Our study focuses on the effect of network structure on epidemic propagation and intervention, and our fixed infection probability fits this purpose well.
Geographical Analysis

References


